AMENDMENTS TO THE CLAIMS:

This listing of the claims below will replace all prior versions and listing of claims in this application.

Claim 1. (**Currently Amended**) A pharmaceutical composition for preventing or treating a Th1-mediated immune disease, which comprises as an active ingredient a substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate in an amount effective to prevent or treat a Th1-mediated immune disease.

Claim 2. (**Currently Amended**) The pharmaceutical composition according to claim 1, wherein the Th1-mediated immune disease is ~~selected from~~ a disease due to graft rejection following transplantation, graft-versus-host disease caused by bone marrow ~~(hematopoietic stem cell)~~ transplantation, ~~and~~ or an autoimmune disease.

Claim 3. (**Currently Amended**) The pharmaceutical composition according to claim 2, wherein the autoimmune disease is ~~selected from~~ autoimmune hepatitis, chronic rheumatoid arthritis, insulin-dependent diabetes mellitus, ulcerative colitis, Crohn's disease, multiple sclerosis, autoimmune myocarditis, psoriasis, scleroderma, myasthenia gravis, multiple myositis/dermatomyositis, Hashimoto's disease, autoimmune hypocytosis, ~~(e.g.,~~ pure red cell aplasia, aplastic anemia [[)]], Sjogren's syndrome, vasculitis syndrome, ~~and~~ or systemic lupus erythematosus.

Claim 4. (Original) The pharmaceutical composition according to claim 3, wherein the autoimmune disease is Crohn's disease or multiple sclerosis.

Claim 5. (Original) The pharmaceutical composition according to claim 1, wherein the substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate is a natriuretic peptide.

Claim 6. (Original) The pharmaceutical composition according to claim 5, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

Claim 7. (Original) The pharmaceutical composition according to claim 6, wherein the atrial natriuretic peptide is of human origin.

Claim 8. (Original) A method for treating a Th1-mediated immune disease, which comprises administering a substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate.

Claim 9. (**Currently Amended**) The method according to claim 8, wherein the Th1-mediated immune disease is ~~selected from~~ a disease due to graft rejection following transplantation, graft-versus-host disease caused by bone marrow ~~(hematopoietic stem cell)~~ transplantation, ~~and~~ or an autoimmune disease.

Claim 10. (**Currently Amended**) The method according to claim 9, wherein the autoimmune disease is ~~selected from~~ autoimmune hepatitis, chronic rheumatoid arthritis, insulin-dependent diabetes mellitus, ulcerative colitis, Crohn's disease, multiple sclerosis, autoimmune myocarditis, psoriasis, scleroderma, myasthenia gravis, multiple myositis/dermatomyositis, Hashimoto's disease, autoimmune hypocytosis, ~~(e.g.,~~ pure red cell aplasia, aplastic anemia [[)]], Sjogren's syndrome, vasculitis syndrome, ~~and~~ or systemic lupus erythematosus.

Claim 11. (Original) The method according to claim 10, wherein the autoimmune disease is Crohn's disease or multiple sclerosis.

Claim 12. (Original) The method according to claim 8, wherein the substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate is a natriuretic peptide.

Claim 13. (Original) The method according to claim 12, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

Claim 14. (Original) The method according to claim 13, wherein the atrial natriuretic peptide is of human origin.

Claim 15. (**Currently Amended**) ~~Use of~~ A method of manufacturing a pharmaceutical composition for preventing or treating a Th1-mediated immune disease comprising admixing a

substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate with a pharmacologically acceptable carrier, excipient or diluent ~~for the manufacture of a pharmaceutical composition for preventing or treating a Th1-mediated immune disease~~.

Claim 16. (**Currently Amended**) The ~~use~~ method according to claim 15, wherein the Th1-mediated immune disease is ~~selected from~~ a disease due to graft rejection following transplantation, graft-versus-host disease caused by bone marrow ~~(hematopoietic stem cell)~~ transplantation, ~~and~~ or an autoimmune disease.

Claim 17. (**Currently Amended**) The ~~use~~ method according to claim 16, wherein the autoimmune disease is ~~selected from~~ autoimmune hepatitis, chronic rheumatoid arthritis, insulin-dependent diabetes mellitus, ulcerative colitis, Crohn's disease, multiple sclerosis, autoimmune myocarditis, psoriasis, scleroderma, myasthenia gravis, multiple myositis/dermatomyositis, Hashimoto's disease, autoimmune hypocytosis, ~~(e.g.,~~ pure red cell aplasia, aplastic anemia [[)]], Sjogren's syndrome, vasculitis syndrome, ~~and~~ or systemic lupus erythematosus.

Claim 18. (**Currently Amended**) The ~~use~~ method according to claim 17, wherein the autoimmune disease is Crohn's disease or multiple sclerosis.

Claim 19. (**Currently Amended**) The ~~use~~ method according to claim 15, wherein the substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate is a natriuretic peptide.

Claim 20. (**Currently Amended**) The ~~use~~ method according to claim 19, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

Claim 21. (**Currently Amended**) The ~~use~~ method according to claim 20, wherein the atrial natriuretic peptide is of human origin.

Claim 22. (Original) A method for regulating the Th1/Th2 balance in the immune system, which comprises treating dendritic cells with a substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine

monophosphate, and thereby polarizing T cells toward Th2-promoting phenotype.

Claim 23. (Original) The method according to claim 22, wherein the substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate is a natriuretic peptide.

Claim 24. (Original) The method according to claim 23, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

Claim 25. (Original) The method according to claim 24, wherein the atrial natriuretic peptide is of human origin.